

estimated glomerulonephritis (14 per cent vs. 29 and 27 per cent) and polycystic kidney disease (6.2 per cent vs. 11.6 and 9.0 per cent). Moreover, we also estimated that on a national scale, blacks would represent 66 per cent of all patients with ESRD due to hypertension, and that 29 per cent of blacks with ESRD would have hypertension as their primary cause. In Jefferson County, Alabama, accelerated hypertension also appeared to be responsible for most of the renal disease seen in blacks between 30 and 39 years of age; interstitial disease, on the other hand, tended to affect blacks over 50.

A greater susceptibility of blacks to ESRD may well account for part of the difference between the rate of referral for ESRD treatment in the United States and the rate in Europe reported by the European Dialysis and Transplant Association (2.2 per 100,000), since Europe has a very small black population. It may also account for some of the regional variation in rates in the United States.<sup>2,3</sup> The greater relative risk of blacks for end-stage hypertensive kidney disease, coupled with the distribution of blacks in the population, may explain why hypertension is one of the major causes of ESRD in the United States, whereas it is of minor importance in Europe.<sup>2,4</sup>

In contrast to the impact of racial composition on rates of treatment of ESRD, sex differences (adjusted for race and age group) were much less important ( $P = 0.0952$ ), although there was some tendency for men to have higher rates than women. Age, however, appeared to be an important factor; we observed a progressive increase in the incidence of ESRD treatment with increasing age and a peak incidence in the sixth decade. Thus, our data suggest that older patients may be more susceptible to the development of ESRD — an important consideration in a country whose population over age 50 is increasing and living longer.

We conclude that the overall rate of entry of new patients into ESRD programs in the United States is higher than originally suggested. We also suggest that rates of new referrals for ESRD treatment programs in a population are influenced greatly by its age and racial characteristics, which may also determine the distribution of causes of ESRD in that population.

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## CHEMICAL DISINFECTION OF CREUTZFELDT-JAKOB DISEASE VIRUS

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**R**ECOMMENDATIONS for the disinfection of virus-contaminated tissues from patients with Creutzfeldt-Jakob disease (CJD), published five years ago in the *Journal*,<sup>1</sup> relied heavily on data from experiments using mouse-adapted scrapie virus — at that time a more conveniently studied member of the same group of spongiform encephalopathy viruses. We have now accumulated sufficient data on guinea-pig and mouse-adapted viral isolates from two patients with CJD to be able to present current recommendations based on studies of the CJD virus itself. Autoclaving for one hour at a temperature of at least 121°C (15 psi) remains the method of choice for the sterilization of contaminated material, but a one-hour exposure to 0.5 per cent sodium hypochlorite (a 10-fold dilution of household bleach) should provide excellent disinfection when autoclaving is not possible.

## METHODS

### Virus Strains

The guinea pig-adapted strain of CJD virus was isolated in our laboratory in primates and guinea pigs from brain tissue obtained at autopsy by Dr. Françoise Cathala (Hôpital de la Salpêtrière, Paris, France) from a 62-year-old woman with a clinical illness and a neuropathological picture characteristic of CJD.<sup>2</sup> Second or third-passage guinea-pig brain homogenate, with a titer of  $10^{5.5}$  to  $10^6$  mean lethal doses ( $5.5$  to  $6 \log LD_{50}$ ) per gram of brain tissue, was

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used in the inactivation mixtures. The mouse-adapted strain of CJD virus was isolated in rats and mice by Dr. Jun Tateishi (Neurological Institute, Kyosho University Medical School, Fukuoka, Japan) and in primates in our laboratory, from brain tissue obtained at autopsy from a 56-year-old man with a prolonged (four-year) but otherwise typical illness, whose brain showed numerous kuru-like plaques in addition to the usual neuropathological changes of CJD.<sup>3</sup> First-passage or second-passage mouse brain homogenate with a titer of 6 to 7 log LD<sub>50</sub> per gram of brain tissue was used in the inactivation mixtures.

### Disinfectants

Bleach, containing 5.25 per cent (w/v [weight/volume]) sodium hypochlorite, was obtained as a commercially sold household product. Crystalline potassium permanganate (J. T. Baker Chemical Co.), formaldehyde solution containing 37 per cent (w/w) formaldehyde, glutaraldehyde solution containing 50 per cent (w/w) glutaraldehyde (both from Fisher Scientific), sodium deoxycholate powder (BDH Chemicals), sodium dodecyl sulfate powder (Pfaltz & Bauer), and Triton X-100 (Research Products International), were all purchased as reagent-grade laboratory chemicals. Chlorine dioxide solution was provided as Alcide by Mr. Howard Alliger, Alcide Corp., Plainview, N.Y. Ethylene oxide was obtained in pressurized gas cylinders from Bel Welding, Beltsville, Md.

### Inactivation Procedure

Ampules of CJD virus-infected 10 per cent or 20 per cent brain homogenate in 0.15 M phosphate-buffered saline, pH 7.4, that had been stored at -70°C were thawed just before use. Equal volumes of brain homogenate and the freshly prepared chemical being tested were mixed to give a final concentration of either 5 per cent or 10 per cent brain suspension and the following final concentrations of each chemical: 10 per cent or 25 per cent bleach, containing 0.5 per cent or 1.3 per cent (w/v) sodium hypochlorite; 0.1, 0.2, or 0.4 per cent (w/v) potassium permanganate; 5 per cent (w/v) sodium deoxycholate; 2 per cent (w/v) sodium dodecyl sulfate; 1 per cent (v/v) Triton X-100; and 50 ppm (v/v) chlorine dioxide, generated from the spontaneous decomposition of chlorous acid in a reaction mixture of 0.036 M sodium chlorite and 0.26 M lactic acid. Incubation mixtures were kept at room temperature and magnetically stirred for the duration of the virus-chemical exposure.

Exposure of guinea-pig brain homogenate to 88 per cent (v/v) ethylene oxide was carried out in an American Sterilizer apparatus operated at 14 psi and 43°C. In experiments using tissue fixatives, terminally ill mice were either perfused with a 10 per cent buffered glutaraldehyde solution containing 5 per cent (w/w) glutaraldehyde, and their brains removed and stored an additional two weeks in glutaraldehyde at 4°C, or they were killed and their brains removed and fixed in a 10 per cent formaldehyde solution containing 3.7 per cent (w/w) formaldehyde for several weeks at room temperature. Brains were repeatedly washed in phosphate-buffered saline during the 48 hours before preparation and inoculation of tissue suspensions.

### Virus-Infectivity Titrations

Aliquots of the virus-chemical mixtures were removed at various intervals after mixing, diluted in 10-fold steps in phosphate-buffered saline, and immediately inoculated into animals, except in the potassium permanganate mouse-passage virus experiment, in which aliquots were mixed with activated charcoal, stirred for 20 minutes, and centrifuged for 15 minutes at 1800 rpm, and the supernatants were stored overnight at -70°C before dilution and inoculation. Guinea-pig-adapted CJD virus was titrated in four-week-old female Hartley strain guinea pigs (Charles River Breeding Laboratories, Wilmington, Mass.), using three 0.05-ml intracerebrally inoculated guinea pigs for each dilution. Mouse-adapted CJD virus was titrated in weanling female Swiss Webster mice (Taconic Farms, Taconic, N.Y.), using six 0.03-ml intracerebrally inoculated mice for each dilution. Animals were observed for signs of disease during a period of 24 months, and brains from a sampling of animals in each dilution group showing typical CJD illness and death were removed and

examined in coded histologic sections for spongiform change. Mortality end points were calculated with the method of Reed and Muench,<sup>4</sup> with titers expressed as log<sub>10</sub> LD<sub>50</sub> per gram of wet brain.

### RESULTS

Data from CJD-virus inactivation experiments are shown in Table 1. Of all the chemicals tested, only sodium hypochlorite produced consistently marked inactivation, decreasing virus infectivity by at least 3 to 4 log LD<sub>50</sub> as early as 15 minutes after initial exposure. Only one animal in a single experiment, inoculated at the lowest tested dilution (10<sup>-1</sup>), showed the clinical signs or histologic spongiform change of CJD.

Chlorine dioxide and potassium permanganate produced only a small degree of inactivation, and the detergents sodium deoxycholate, sodium dodecyl sulfate, and Triton X-100 had virtually no effect. End-point infectivity titrations of glutaraldehyde and formaldehyde-fixed brain tissue were not done; however, as judged from the incubation times of mice dying after inoculation with 10<sup>-1</sup> tissue dilutions, glutaraldehyde produced only partial inactivation, and formaldehyde had no effect.

### DISCUSSION

Although sodium hypochlorite (household bleach) is the most consistently effective chemical disinfectant of the CJD virus, it has the practical disadvantage of being corrosive to skin, fabrics, and certain metals. Chlorine dioxide has produced moderate to substantial inactivation of scrapie virus in previous experiments using the same 50-ppm concentration employed in the present study,<sup>5,6</sup> and it is possible that doubling or tripling this concentration would be more effective against the CJD virus. Potassium permanganate has also produced variable to complete inactivation of scrapie virus<sup>6-8</sup>; our data suggest an increasing, if still moderate, inactivation of CJD virus with increasing chemical concentrations, and experiments are now in progress with concentrations as strong as 1 per cent instead of the customary 0.1 to 0.05 per cent solutions used for general antimicrobial disinfection. The potential advantage of using these chemicals lies in their complete lack of corrosive properties and toxicity in human beings.

Certain other disinfectants that have been studied in experiments using scrapie virus were not reexamined in the present study. Iodine and hydrogen peroxide have not shown enough disinfectant activity against scrapie virus to warrant testing against CJD virus,<sup>6</sup> and more active chemicals such as periodate and phenol are impractical, in that periodate inactivation must be carried out in the dark, and phenol is only effective at nearly saturated concentrations, which are unacceptably toxic for use in disinfection in hospitals.<sup>9,10</sup>

Under the best of conditions, it may be asked whether chemical disinfection can ever be considered complete, or whether there may exist some small amount of residual viral infectivity after treatment. The answer

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will depend in part on the level of infectivity in pre-treatment tissue, but because of the expense of the most sensitive assay — titration in primates — such information is now limited to data on 17 cases referred to our laboratory, from which serial dilutions of brain tissue were inoculated. In most cases, disease transmission did not occur after inoculation of 0.03-ml volumes of dilutions higher than  $10^{-3}$  to  $10^{-4}$ , and in no case after dilutions higher than  $10^{-5}$ . Enough animals were inoculated in a few cases to permit calculation of Reed-Muench infectivity titers, which ranged between 3 and 6 log LD<sub>50</sub> per gram of original brain tissue. CJD virus-infectivity levels in peripheral tissues are almost certainly lower than they are in the brain, judging from studies of virus titers in tissues of animals with natural and experimental scrapie.<sup>11,12</sup>

These infectivity levels in human tissue are comparable to levels in the guinea-pig and mouse-passaged lines of CJD virus used in the present study, and they are substantially lower than those of the mouse or hamster-passaged lines of scrapie virus used in earlier inactivation studies. Protection of all but one animal inoculated with even the lowest dilution of hypochlorite-treated CJD virus-infected brain tissue, which before treatment had infectivity levels of at least 5.5 log LD<sub>50</sub> per gram, thus suggests the possibility of achieving complete disinfection of most contaminated human tissue.

The preferred method of disinfection — a one-hour exposure to either 0.5 per cent sodium hypochlorite or autoclaving at a temperature of at least 121°C — will depend on the material to be treated. Chemical decontamination is obviously the only means that can be used for large or fixed surfaces, such as operating or autopsy tables, workbenches, and floors. Autoclaving is necessary for fabrics and for delicate or corrodible items, such as metal instruments, microtome knives, or even small microtomes themselves. For contaminated material that can withstand either chemical or autoclave treatment — for example, glassware, rubber gloves, plastic containers, and all disposable items — the autoclave alone or chemical treatment followed by autoclaving is the preferred method of decontamination.

Finally, it is well to reemphasize that CJD virus has yet to be isolated from body surfaces, secretions, or excretions, and it has been found only irregularly in non-neural peripheral tissues from human beings<sup>13</sup>; in addition, numerous epidemiologic studies published in the past few years have shown that none of the people in closest contact with patients with CJD (wives, friends, employee contacts, members of the medical or nursing professions, or paramedical personnel) appear to have a higher risk of contracting CJD infection than does the general population.<sup>13-15</sup> It is also reassuring that not a single case of CJD has yet been reported to occur in workers most exposed to infective tissues from patients with CJD (neuropathologists, research scientists, and laboratory personnel),

Table 1. Inactivation of CJD Virus-Infected Guinea-Pig or Mouse-Brain Suspensions after Various Periods of Exposure to Chemicals.

CHEMICAL	FINAL CONCENTRATION	PERIOD OF INCUBATION (minutes)					
		15	30	45	60	180	240
Decrease in LD <sub>50</sub>							
Sodium hypochlorite *	0.5% (w/v)	≥3.5	3.5		≥3.5		
Sodium hypochlorite *	1.3% (w/v)		≥3.5				
Sodium hypochlorite †	1.3% (w/v)		≥3.8				
Chlorine dioxide *	50 ppm (v/v)	1.3	1.2		2.3		
Potassium permanganate *	0.1% (w/v)	1.3	0.8		1.3		
Potassium permanganate †	0.1% (w/v)	0	0	0	0		
Potassium permanganate †	0.2% (w/v)	<0.5	<0.5	<0.5	<0.5		
Potassium permanganate †	0.4% (w/v)	1.1	1.8	3.1	2.0		
Ethylene oxide *	88% (v/v)				1.0	1.0	0.5
Sodium dodecyl sulfate *	2% (w/v)						≤1.5
Sodium dodecyl sulfate †	2% (w/v)						0
Triton X-100 *	1% (w/v)						≤1.5
Triton X-100 †	1% (w/v)						0
Sodium deoxycholate *	5% (w/v)						1.0
Sodium deoxycholate †	5% (w/v)						0
Formaldehyde †	3.7% (w/w) (fixed brain, no inactivation ‡)						
Glutaraldehyde †	5% (w/w) (perfused brain, partial inactivation ‡)						

\*Incubated with guinea-pig-adapted strain of CJD virus with a titer of 5.5 to 6 log LD<sub>50</sub> per gram of brain tissue (three animals per dilution point).

†Incubated with mouse-adapted strain of CJD virus with a titer of 6 to 7 log LD<sub>50</sub> per gram of brain tissue (six animals per dilution point).

‡Estimation made from comparison of incubation times in groups of six animals inoculated with  $10^{-1}$  dilutions of the treated or untreated virus-infected control brain suspensions.

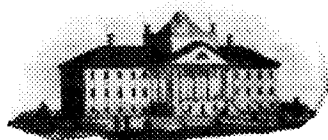
or in nonhuman primates that for periods of up to 10 years have been continuously exposed to experimentally infected animals in our primate colonies.<sup>16</sup> Thus, despite proved person-to-person transmissibility of the disease by invasive procedures,<sup>17,18</sup> the risk of acquiring CJD by any other means than tissue penetration by contaminated material must be very small indeed.

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## CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL



### Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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### CASE 21-1982

#### PRESENTATION OF CASE

A 30-year-old man was admitted to the hospital because of multiple trauma.

He was apparently well until the evening of admission, when he was involved in a motorcycle accident. He was taken to another hospital, where physical examination showed that the pulse was 84 and the blood pressure was 60/30 mm Hg. The patient was alert and oriented. Multiple fractures were noted. The abdomen was distended and tender. An x-ray film of the chest showed widening of the mediastinum. Nasal oxygen was administered, and a nasogastric tube and an indwelling bladder catheter were inserted. Fluids were administered intravenously, followed by a transfusion of 2 units of whole blood. A nasotracheal tube was passed, and military antishock pressure trousers were applied. The blood pressure rose to 95/50 mm Hg, and the patient was transferred to this hospital. During his transportation via ambulance the blood pressure fell to 40 mm Hg systolic, although he remained alert.

On arrival in the Emergency Ward of this hospital the patient was alert and responsive. The systolic blood pressure was 50 mm Hg. He moved both upper extremities. The pupils were equal and reactive, and no evidence of head injury was observed. The lungs and heart were normal. The abdomen was distended and diffusely tender; bowel sounds were absent; no organs or masses were felt. There was evidence of fractures of both tibias and of the left femur.

The hematocrit was 33.4 per cent, and the platelet count 19,000. Another x-ray film of the chest (Fig. 1) revealed that an endotracheal tube ended 1 cm above the thoracic inlet and a nasogastric tube terminated within the stomach; a screw was projected over the left upper portion of the abdomen at the base of the lung; a right subclavian line was sharply kinked and crossed to the left brachiocephalic vein; the superior mediastinum was widened, and the aortic knob was indistinct; the lungs and heart appeared normal.

A left subclavian line was inserted, fluids were given intravenously, and two transfusions of group O, Rh-negative packed red cells were administered. Tetanus toxoid was injected intramuscularly, and an anti-G suit was applied. No urinary output was observed. The blood pressure rose to 100 mm Hg systolic. The patient was taken to the operating room, the anti-G suit was deflated, and an exploratory laparotomy was performed 30 minutes after his arrival. The peritoneum was entered, and 2 liters of fresh blood and clots were evacuated. The liver and spleen appeared intact, and no rapid reaccumulation of blood was evident. Examination of the small bowel showed a rent in the mesentery of the distal ileum. Several arteries that were bleeding actively in that region were clamped and ligated, and the contiguous portion of the ileum, which appeared nonviable, was resected. A small perforation of the proximal ileum was repaired. Scattered subserosal hematomas were seen in the remainder of the small bowel. A tear of the colonic serosa was closed. A hematoma in the right side of the pelvis, from which small amounts of blood oozed through a tear in the overlying peritoneum, appeared stable during a period of obser-